

Statement from the Work Session on Environmentally Induced Alterations in Development: A Focus on Wildlife

In the last five decades many wildlife populations across the North American continent have experienced severe and often sudden declines that in some cases have led to extirpation. While some of these declines are attributable to natural biological events such as epidemics caused by microbial pathogens and well-established human causes such as overharvesting, habitat changes, and introduction of exotic species, many of these declines have not been satisfactorily explained by these factors. During this time a number of man-made compounds were introduced into the environment that caused the declines of some wildlife populations through adult and embryonic mortality.

Many man-made contaminants can alter embryonic and early postnatal development. The consequences of this interference are irreversible, in some cases leading to early death, but in other cases not manifested until the individual reaches adulthood with resultant loss of fertility. Through subtle biochemical and physiological changes, the contaminants interfere with the development of the reproductive, endocrine, immune, and nervous systems of embryos, and therefore are likely candidates for causing some of the declines and failure of wildlife populations to recover even after regulatory prohibition of activities involving these compounds. In light of this knowledge, 23 wildlife experts were gathered in retreat at Wingspread, Racine, Wisconsin, 10–12 December 1993, to discuss the topic of "Environmentally Induced Alterations in Development: A Focus on Wildlife." Participants reported

on their observations of alligators, bald eagles, beluga whales, boreal toads, bottlenosed dolphins, Caspian terns, common terns, Florida panthers, Forster's terns, great blue herons, herring gulls, leopard frogs, old squaw ducks, polar bears, roseate terns, scoters, sea turtles, slider turtles, spectacled eiders, water fleas, white croakers, and wood ducks. Participants were expected to reach some conclusions concerning the nature, magnitude, and geographic scope of the problem on the North American continent.

The following consensus was reached by participants at the workshop.

1. We are certain of the following:

Declines in a number of species and many taxa (including plants) are in progress on the North American continent. Some of these declines are related to exposure to man-made chemicals. Such declines are not solely a U.S. or North American problem but are occurring on a global scale.

There is a special cause for concern for long-lived species which may or may not (at this time) show overt signs of reproductive impairment. Examples of species that are near extirpation at the population level are those that are annually replenished by outside stock, not by intraregional reproduction, such as Great Lakes lake trout and shoreline bald eagles, and Lake Apopka, Florida, alligators and turtles.

Populations of many long-lived species are declining, some to the verge of extinction, without society's knowledge. The presence of breeding adults and even healthy young does not necessarily reflect a healthy population. Detailed population analysis is needed to determine whether offspring have the functional capacity to survive and reproduce.

Wildlife are exposed to compounds that disrupt development of the reproductive, immune, nervous, and endocrine systems and thereby can lead to population instability. The pollutants of greatest concern affect cellular and molecular processes that regulate developmental, endocrine, and immunological functions. Hormones are natural substances that control normal

development of all embryos and fetuses. Many of the contaminants mimic or interfere with female and male hormones, thereby modifying development and reproduction.

The embryo is the most sensitive life stage of animals to the hazards posed by these chemicals.

Current contamination in wildlife has reached levels in some regions at which there are known sublethal effects sufficient to impair populations. Unless the continuing release of man-made toxic chemicals is prohibited immediately in certain well-studied North American major aquatic systems, populations of important top predator species may become extirpated.

Many persistent contaminants do not remain at the site of release. Chemical releases on one continent may not only affect animals on that continent, but also animals on other continents and in other hemispheres. They are carried as particulates or gases in the air, surface waters, groundwater, and ocean currents across or between continents and by animals that travel long distances from the site of contamination. The contaminant, therefore, can enter the food web in places remote from the site of release.

It is no longer sufficient to approach population/species revitalization passively by providing appropriate habitat and expecting threatened or extirpated populations to recover. Contamination of apparently useful habitat is not always visible and may not cause overt lethality. Instead, contaminants may cause population-threatening changes in functionality. For example, populations may not be able to recover from *a*) infectious diseases because of immunosuppression; *b*) the inability to obtain sufficient food, avoid predators, and the loss of parenting instinct because of neurotoxicological effects; or *c*) the result of abnormal sexual development of anatomy or behavior because of endocrine disruption.

It has been difficult to document causal relationships between population declines and failures with the chemicals suspected to have caused them. The difficulty can only be successfully addressed through

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multidisciplinary research linking ecological, wildlife, human, and laboratory animal research and by building bridges between the human, veterinary, and environmental health sciences.

Increased collaboration and exchange of information between wildlife, laboratory animal, and human investigations is essential. This requires a redefinition or broadening of the role of federal agencies and the institutionalization of causal investigations (environmental detective work). This work must be supported by federal agencies to investigate the declines and their causal relationships.

Links have been made between some effects and specific man-made chemicals. Other chemical-effect linkages, which in fact may exist, have not been made for several reasons because *a*) it is difficult to distinguish individual effects of the numerous man-made chemicals present in the environment because many have similar biological effects; *b*) other biotic and abiotic factors play a role in population instability; *c*) testing for the presence of such chemicals is so expensive; and *d*) additional research is required to clarify these connections (see below).

Currently, regulatory action depends upon knowledge of the specific physiological or biochemical end point of a chemical. However, we have yet to identify many biomarkers that are specifically diagnostic of a substance or an effect. Because few such biomarkers have been developed, there is clearly a need for more physiological and molecular-oriented, biomarker based research. However, we believe that the prerequisite of documenting the mode of toxic action and development of specific biomarkers to make cause and effect links before taking regulatory action leads to unnecessary delays that continue to result in injury and potential extirpation of more populations and species.

The issues addressed in this statement are fundamental to the concepts of biodiversity, sustainable development, and ecosystem health and call for responsible global citizenship. They assume a preemptive, rather than a reactive, role for scientists, wildlife agencies, and environmental regulators (see below).

2. We estimate with confidence that:

In many cases wildlife and humans have exceeded their capacity to compensate for exposure to chemicals.

As more areas of the North American continent are surveyed and a wide variety of multigenerational consequences are taken into consideration, more evidence of

damage will be revealed among wildlife and human populations. Many effects are taking place that are not easily observed, but they do exist. Too much attention has been directed toward the health of the current population, the directly exposed individuals, and not enough on the offspring. Our current regulatory focus on adult mortality is not sufficient to preserve reproductively successful populations. Rather, we must ensure that successive generations can reproduce.

When an animal is exposed at the same time to many chemicals that individually are at nontoxic levels, additivity, antagonism, potentiation, and synergy can result in unpredictable consequences. Concomitant exposure to multiple chemicals can cause massive or subtle, but potentially tragic, effects.

When chemically induced syndromes have been identified in the past, it has been difficult to establish cause and effect relationships, and to do so has frequently taken decades. The basic tenets of epidemiology have proved valuable in organizing information to infer causal relationships. The tenets include *a*) time order (exposure must precede the effect); *b*) strength of association (relative risk); *c*) specificity of a compound to an effect (does X lead to Y?); *d*) consistency on replication (results are supported across studies, geographic areas, and over time); *e*) coherence with biological theory (the relationship must be biologically plausible); and *f*) performance on prediction (does the test stand up in the field?).

Wildlife are reliable sentinels of effects of chemicals on human populations. Lesions at every level of biological organization provide parallels and excellent models for assessing the response of humans exposed to the same chemicals. Effects seen at the cellular levels to the population levels among wildlife populations should be an integral part of assessments of risk or injury to human health.

3. There are many uncertainties in our predictions because:

The implications of cumulative exposure to the vast number of chemicals released in the environment are difficult to determine. Although data may become available on the mechanisms of action of certain chemicals, there is no way to account for the interaction of the large number of chemicals to which an animal is exposed.

Regional differences in land-use practices, industrial activity, and geophysical characteristics must be factored into a

cumulative, multimedia exposure model. Migratory species pose additional difficulties when determining sources of exposure.

No systematic, coordinated effort has been undertaken to determine the geographic extent to which contaminants contribute to the degradation of wildlife populations. While there is evidence in areas where researchers have suspected and looked for wildlife damage, there are many more areas of the continent that have not yet been surveyed. Consequently, the magnitude of the damage is incompletely understood for major wildlife taxa.

U.S. and Canadian governmental agencies no longer appear willing to commit sufficient long-term, fiscal resources for documenting the effects of chemical exposure in wildlife. Until responsible parties are designated and given adequate appropriations to address wildlife health problems, biodiversity will continue to decline.

4. We believe that:

Traditional assessments of risks posed by single chemicals are not adequate for assessing the risks for embryos exposed to multiple chemicals.

The tenets of epidemiology have been successfully used to infer causal associations between certain syndromes and exposures to multiple chemicals.

Model systems for characterizing wildlife population declines can help to determine the magnitude and scope of the problem. Initial efforts must concentrate on a suite of contaminants thought to have the most serious consequences. It is imperative to replicate the damage observed in the field under laboratory conditions to confirm cause and effect linkages.

All chemicals licensed for environmental release should be tested throughout a minimum of two generations for a wide variety of effects including reproductive, immunological, endocrinological, and neurological end points.

To abate the scope and severity of wildlife population declines, we must endeavor to bring the message into our homes, schools, and the political arena. Until more people understand the insidious nature of developmental toxicants, little will change. More popular press articles and other media should broadcast the message about the effects of developmental toxicants using the wildlife/human connection. A major popular press publication is needed to get the message to the public. Pressure must be exerted on legislators and public health officials to take action to restore damaged populations and to prevent further damage from occurring.

5. To improve our assessment of dangers posed by chemicals:

A balanced and comprehensive assessment of wildlife declines and diseases caused by chemicals on a global basis is imperative. These assessments must include both retrospective as well as prospective information.

Funding must be forthcoming to support interdisciplinary investigations, the use of nontraditional organisms (such as wildlife), and research that focuses on functionality in addition to obvious damage. It is imperative to establish a central coordinating office and interdisciplinary teams that can report on sites where subtle effects are occurring and be responsible for directing where field samples can be shipped for initial and rapid evaluation of effects and identification of suspected chemical causes.

An electronic network should become available to wildlife biologists to improve networking and to increase opportunities for collaboration. In light of the strenuous and unpredictable nature of field research, networks could increase the maximum utilization of sampling and the power of each study.

There is an urgent need to have integrated funding mechanisms established to facilitate these studies, possibly by creation of a national institute of the environment.

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This consensus statement reflects the professional wisdom of the scientists at the work session and not necessarily the institutions or agencies in which they are employed.